LESSONS LEARNED FROM A BLIND SCHOOL SURVEY

by

Robert J. Masters


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I have used this culture method after that experience in all cases. And we hold everybody in quarantine until we get rid of some of them as carriers. I maintain the quarantine in this way; those in high school I send back immediately; those in the junior school I let go back soon, realizing that the incidence was not high in those schools. It is in the lower grades. The children in the lower grades we hold until they have their tonsils out, until the school ends, or they become negative. Finally all of them go back and then nothing happens. Our scarlet fever epidemic ends and as summer comes there is not a case left.

I feel that with culture control you can do a lot, and with a protraction of the quarantine where necessary, rather than an arbitrary general shortening or lengthening of quarantine, you can prevent convalescent carriers. It does seem that these streptococcic germs become avirulent, or that they are subject to mutation. I think the virulence goes up to a certain point and then goes down, but I am not sure of that.

The Doctor will have to prove conclusively to me that these diseases he discusses are interchangeable. I think there are conditions that he has described that probably cause the mutability.

I think we should have one more thing to add to our control and that is this: When I get a negative or two negatives or three negatives, will Dr. McShane let me turn the case loose in three weeks? He has refused so far. He has said, "no shortening of the four-week period."

Dr. John J. McShane: Dr. Bailey, in your study of the different diseases that you have reported on, did you find more than one type of streptococci in this group? If there is a large number of varieties of hemolytic streptococci, I would like to be advised whether you found more than one, two or three strains among this group.

LESSONS LEARNED FROM A BLIND SCHOOL SURVEY

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Four years ago a statement was encountered to the effect that many State Schools for the Blind have very inadequate and inaccurate records of the causes of blindness of their students. This almost incredible charge led to an investigation of the records at our own Indiana State School for the Blind. Only about five minutes of study were required to determine that no records could be worse than those of our school. A few examples of the causes of blindness given on the entrance applications of the students, together with the causes determined by examination, are shown in the following list.

CAUSES OF BLINDNESS

TABLE 1 Given on Application	TABLE 2 Found by Examination
1. "Following flu."	1. Buphthalmos.
2. "Do not know."	2. Ophthalmia neonatorum.
3. "As near as we know, skull fracture."	3. Hereditary macular degeneration. Optic atrophy.
4. "Chicago specialists could not determine."	4. Albinism with macular degeneration and optic atrophy.
5. "Right eye, trauma. Left eye, congenital amblyopia."	5. Right eye, corneal scar, reads Jaeger No. 3. Left eye, syphilitic chorioretinitis with extensive scars.
6. "Right eye, tumorous condition. Left eye, cataract."	6. Right eye, persistent tunica vasculosa lentis with mal-development of iris. Left eye, uveitis with cataracta complicata.
7. "Fall on the head plus blood disease."	7. Luetic disseminated chorioretinitis with optic atrophy.
8. "Neglect."	8. Ophthalmia neonatorum.
9. "Born blind."	9. Congenital cataract.
10. "Inheritance."	10. Congnital aniridia and upward dislocation of lenses.
11. "Eye-Ritus."	11. Interstitial keratis and uveitis.
12. "Doctor used too-strong drops in the eyes at birth."	12. Ophthalmia neonatorum.

With the firm belief that no study could be more interesting than that which was greatly needed and long overdue at the school, a systematic survey of the student personnel was undertaken and has only recently been completed. Many diagnostic problems were encountered, which are deserving of comment. Several lessons were learned, which should arrest the attention of every ophthalmologist.

The diagnostic difficulties were dependent upon several factors. One of these was the complete lack of an adequate history. Nothing more than the foregoing chart is required, to give a vivid understanding of the kind of histories which were available in writing. The histories given by the students at personal interviews were indefinite and valueless. Thus nothing remained but to reach a conclusion, as to the cause of the blindness of each eye, by examining it externally and ophthalmoscopically. Fear of the examination disturbed some of the students, and made thorough inspection difficult. Rapid nystagmoid movements of the eyes, or inability to fix the

Read before Section on Eye, Ear, Nose and Throat, Illinois State Medical Society, Peoria, May 18, 1937.

gaze, made of the ophthalmoscopic examination a challenging task, especially trying since most of the inspections were made through undilated pupils. The abstinence from use of mydriatics was the result of a promise made to the superintendent in office at the time the examinations were first undertaken. Our present superintendent, Mr. Robert Lambert, has been most cooperative, and under his regime there have been absolutely no restrictions to hinder satisfactory work.

With no histories and only objective findings to aid in differential diagnosis, the resulting problems were many. For example, the eyes of two chldren exhibited pictures quite typical of the sequellae of ophthalmia neonatorum. One of these, under careful observation since she was ten days old, has never had a conjunctivitis. She was born with corneas which had failed to clear, plus microphthalmia and sclerotics congenitally thin. The other child had a definite history of destructive corneal ulceration which complicated scarlet fever at five years of age, with normal eyes prior to that time. Without histories, both of these diagnoses might have been wrong. Even so, without histories on most of the students examined, there may be several diagnoses wrongly determined and recorded in the table to follow shortly.

After arriving at reasonably accurate determinations of the causes of blindness, their classification presented further difficulty. Correct grouping of the large number of optic nerve atrophy cases was hindered by frequent doubt as to their underlying pathology. Furthermore, many of the eyes suffering from aberrations in development exhibited many different anomalies: congenital cataract or coloboma of the iris were sometimes associated with microphthalmia; congenital coloboma of the macula showed an accompanying optic atrophy; and one pair of eyes exhibited congenital cataract, optic atrophy and macular degeneration. The classification which was finally evolved is listed in the following table:

TABLE OF CAUSES OF BLINDNESS

(152 Students Examined)

Cause	Number	Percentage
1. Optic atrophy		
a. Associated with congenital or hereditary retinal maldevelopment or degeneration	15	
b. Postneuritic	10	
c. Assoc. with luetic chorioretinitis	9	
d. Assoc. with congenital macular coloboma	5	
e. After meningitis	5	
f. Associated with pigmentary degeneration of the retina	3	
g. Cause not determined	2	
Total	49	32.2%
2. Congenital cataract (Some with dislocations, microcornea, O.A., retinal degeneration, etc.)	31	20.4
3. Ophthalmia neonatorum	21	13.7
4. Uveitis (with sequellae)	11	7.2
5. Buphthalmos	10	6.5
6. Interstitial keratitis Three sequellae of uveitis, one with O.A.	10	6.5
7. Congenital aniridia	4	2.6
8. Trachoma	3	1.9
9. Sympathetic ophthalmia	3	1.9
10. Coloboma of iris, ciliary body and choroid, microphthalmos	2	1.3
11. High myopia	2	1.3
12. High hyperopia	1	0.65
13. Corneal ulceration with scarlet fever	1	0.65
14. Microphthalmos, congenitally opaque corneas	1	0.65
15. Neuroepitheliama (both eyes enucleated ten years ago)	1	0.65
16 Albinism	1	0.65
17. Cause undetermined (examination not permitted)	1	0.65

The interesting ophthalmological survey of the Illinois State School for the Blind, reported in the July, 1934, issue of the *American Journal of Ophthalmology* by Adams, Gamble, Gifford and Gradle, revealed the fact that approximately 25% of the students in the school belonged in sighted classes. Of these, 7.5% belonged in public school, and 17.2% were candidates for sight-saving classes. The close approximation of these figures to the findings at the Indiana State School is immediately apparent upon examination of the following chart. (The ability to read Jaeger types, upon which many of the visual acuity estimates were made, may be open to criticism. However, this test would seem to give a fair idea of the students' ability to acquire sighted or partial-sighted training.)

Students who need blind school training	114	75%
Students who should be in sight saving classes. (J. 12 to J. 7 inclusive at 20 cm.)	33	21.7%
Students who should be in public school (J. 6 or better at 20 cm.)	5	3.3%

In a second communication (A. J. O., January, 1937), Adams, Gamble, Gifford, and Gradle, reported a reduction of the students who should be in public school to 3.1%, with all of the

17.2% who needed it, receiving sight-saving class instruction at the school. This is an excellent economic and social contribution, which we are still lacking in our Indiana School.

Not long ago the monthly meeting of the eye staff of the Indiana University School of Medicine was held at the School for the Blind. At this meeting a large group of students was exhibited, some because their eyes presented problems in diagnosis and others because they represented interesting aberrations in development. Some of the men commented about the remarkable museum of pathology which they had been privileged to study. Others were depressed by the utterly hopeless condition of the large number of eyes which they had seen. But the greatest number of them remarked with deep feeling about the large number of blind eyes which should not have been blind. This thought is the one which most arrestingly and depressingly overshadows all others, when any large group of blind children is examined. The blindness of so many of them is so unnecessary and could have been so definitely prevented.

The children in our school with preventable blindness have been tabulated as follows:

1.	Ophthalmia neonatorum	21 Cases	
2.	Interstitial keratitis	10 Cases	
3.	Uveitis, at least some of the	11 Cases	
4.	Optic atrophy with luetic chorioretinitis	9 Cases	
5.	Trachoma	3 Cases	
6.	Sympathetic ophthalmia	3 Cases	
7.	Congenital cataract (birth control)	9 Cases	of 31
8.	Congenital aniridia (birth control)	3 Cases	
9.	Buphthalmos	10 Cases	
	Total	79 Cases	

There seems to be deeper irony in the fate of these youngsters when we note how very profound is the visual loss of some of these very children who should not be blind at all, those with the sequellae of ophthalmia neonatorum, sympathetic ophthalmia, buphthalmos and leutic optic atrophy being vivid examples. No child should have interstitial keratitis. It is reasonable to believe that most cases of uveitis should be saved from blindness by early, adequate and persistent treatment. A woman may reasonably expect to have children without congenital cataract (unless she and her husband are both so afflicted themselves, as were the parents of two of our students). But if she deos have one child with congenital cataract, why should she have four, or three, or two with this condition? In the group of nine preventable congenital cataract cases, listed in the above table, only those *after* the first one in each family are included.

It is this large percentage of children with preventable blindness which hurls an unavoidable challenge to the ophthalmologists. What are we going to do about it? How can we give the best service in this branch of preventive medicine? Very little thought leads to the firm conviction that we can do very little by ourselves. We may work our hearts out in the effort to save eyes already on fire with gonorrheal ophthalmia, uveitis or sympathetic ophthalmia when they come under our care. But before we come into the picture someone should have known better than to have allowed these conditions to occur. Some of us, who are privileged to work with groups of blind young people, find several of them who are anxious to discuss the question of their marrying and having children. But our opportunities are few for advising young people about their prospective parenthood.

Further thought soon leads to the conclusion that our fellow physicians and the social service workers are the two groups upon whom we must depend for our help in this work. As in other branches of medicine, there are many phases of ophthalmology which depend for their successful management as much upon competent, well-trained social service workers as they do upon the physicians themselves. Any physician who has enjoyed the cooperation of a well organized social service department will agree with this statement. Our cooperation with the social workers should be unreserved, careful and conscientious, whenever it is asked of us.

Of all the lessons learned during this survey, the most impressive is how utterly dependent are we ophthalmologists upon our fellow physicians, for aid in the prevention of blindness of children. We are often asked, by our local medical societies, to prepare papers for their programs. No subject should be of more interest or value to them than the prevention of blindness. It is in fact our duty to periodically bring this subject to them. We should remind them of the importance of routine prenatal Wassermann tests upon their expectant mothers, with treatment of mother and child as indicated. Interstitial keratitis and chorioretinitis of congenital lues can be

thus eliminated. We should review with them the correct application of Credè treatment to new-born babies' eyes. Silver nitrate conjunctivitis, when it occurs, need not be embarrassing and it does not blind babies' eyes. Careful routine observation of the eyes of children during attacks of febrile disease, with determination of the exact cause of all "red eyes," may prevent blindness from the effects of untreated uveitis. All men engaged in obstetrics and pediatrics, either as specialists or in connection with their general practice, should know how to use an ophthalmoscope well enough to discover opacities in the media and gross lessions in the fundus. The family doctor, discovering the presence of congenital cataract in the first child, can intelligently and logically explain to the parents how great is the possibility of congenital cataract in any further children they may have. His check on the size of the globe, the diameter of the cornea, may bring the child with buphthalmos to the eye surgeon in time to save useful vision. We should talk about these things to our colleagues in other branches of medicine until they get tired of listening to us.

As a concluding thought, would it not help in the prevention of blindness if each one of us reported to the proper organization every new case of blindness, in child or adult, occurring in our own practice every year? In Illinois, your own Society for the Prevention of Blindness should receive these reports. In Indiana we have no such State organization as yet, but the National Society would eagerly welcome such valuable data. To this suggestion the objection has already been raised that much duplication would occur, as these blind people go from one doctor to another in search of aid. Giving the patient's name would avoid this, as would also our definite adherence to the rule of reporting only those patients who lost their vision while under our own treatment.

23 East Ohio Street.

DISCUSSION

Dr. Austin A. Hayden, Chicago: I want to thank Dr. Masters for the tabulation he made in Article 4 of the Chicago Survey. The article reads that the Chicago specialists could not determine the cause—and he very cautiously skipped that part. Seriously, Dr. Masters has given a very comprehensive resume of the situation in Indiana. I noted his figures, and of 79 cases cited, as he says a very large number were in the preventable class, 21% could have been saved by silver nitrate properly instilled. I regret that he did not state how many of these were physicians' cases and how many were midwives' cases or unattended.

In the matter of interstitial keratitis and optic atrophy, it is a strang thing, but it is a fact that of all the progress made in medicine since the Wassermann test was introduced for syphilis, only a very few hospitals in the country have a Wassermann blood test, to say nothing of spinal fluid test, made routinely in all obstetric cases. I had occasion to call attention to this not long ago before the American Medical Association Section on Ophthalmology, and I found that ophthalmologists were not very keen about the observation of this, and that obstetricians were almost in the same classification. I am told that a Wassermann-negative mother cannot give birth to a syphilitic child, and that even if the disease is discovered as late as the third month of pregnancy, a case of interstitial keratitis rarely happens.

I was impressed by what Dr. Masters said about the value of the social worker. Miss Audrey Hayden was responsible about three weeks ago for a broadcast over one of the large Chicago radio stations, on Sunday at twelve o'clock, and I heard this while riding through Indiana, and it had to do with the same subject of building better citizens. It was put on by some association in Chicago, and very dramatically presented the thought you expressed when you said the social worker has a place in this campaign. Let me say that the moving picture of syphilis that is being produced by the United States Public Health Service and the American Medical Association, will have, I hope, a very stimulating effect in making for a much more active campaign on the part of all doctors, public health, private and clinic, toward the elimination of syphilis. Of course nobody things syphilis will be eliminated, but the effort is toward the reduction of the prevalence of syphilitic disease.

Dr. Philip Corboy, Chicago: I would like to ask one question, not dealing with the fact that the mother can be tested. It has been my impression that pediatricians differ as to the value of a Wassermann or a Kahn in the child, and it has been my experience that neither has any significance in the first six months of life in making serological diagnosis.

Dr. C. W. Hawley, Chicago: I am not competent to discuss the statistics of blindness, but I can discuss the question of ophthalmia neonatorum. I do not believe a single child need lose an eye from this cause. That is a broad statement. I had a clinic in Cook County Hospital for many years, and of course we had many cases there, and in all the cases I saw I never lost an eye or had a blind eye. I learned how to take care of ophthalmia neonatorum, and I believe I can say that not a single baby need lose its eye if taken care of properly. This is not the place to tell how to do it, and I will not discuss that, although I have done so before. What I say is true, as you may know if you

believe my reputation for truthfulness and veracity. I had a large clinic at the Post-Graduate Hospital and here too a good many babies were brought in with opthalmia neonatorum. We had some cases of gonorrheal ophthalmia in older patients, and these cases were taken care of in the same way with very satisfactory results.

Dr. Robert J. Masters, Indianapolis (closing): I wish to thank all the discussors, particularly Dr. Hayden for emphasizing the great things that can be accomplished in the way of prevention of those forms of blindness which are dependent upon congenital lues. I only included the "specialists not able to determine" case, because it was one of the funny causes of blindness that came in, and of course there was no way of telling that an eye physician had really seen the child in question.

Answering the question as to early Wassermann, it is my impression that during the early months of infancy the Wassermann test is not as reliable as we would wish. This emphasizes the importance of prenatal tests in the mother. When the tests are so readily available these days, free if necessary by the State laboratories, they should be routinely made.

In outlining the things to discuss with men in general practice at home, I only tried to touch upon a few of them. I believe it is interesting to a general medical society to hear a talk on the causes of blindness as they relate to the cases in which men in their own practices may help in the prevention of blindness.

SOME DANGERS OF RAPID DIURESIS

M. Herbert Barker, M. D.

CHICAGO

The literature abounds with articles on diuresis, but very little is said about certain dangers of rapid diuresis in that great group of cardiac, renal and renal-vascular disease which makes up our greatest number of cases of edema. With the improvement of diuretic management, edema as such is much more readily controlled and we are too frequently tempted to watch the urine volume curve or weight drop without sufficient regard to the concentration of the waste products which may be less easily eliminated. Certainly, during diuresis, the piling up of minerals or medication with a resultant acidosis, uremic state or drug poisoning readily occurs.

Diuresis has always been a problem to the physician. The patient is usually more disturbed over the external evidences of a disease, so that a gross anasarca may instigate more vigorous therapy than a more serious disease which he is unable to observe. With the remarkable report on Foxglove by William Withering[1], came one of the greatest steps for relieving the patient of cardiac dropsy. Thus, digitalis opened a whole new chapter in pharmacology which still advances. The great array of zanthine compounds clearly show the continued demand for better diuretics. the same may be said of the mercurials. The use of calomel in the edema of nephritis appears as a regular practise in the protocols of Richard Bright.[2] Saxl,[3] Keith,[4] Barker and O'Hare[5] and others have written on the efficiency of the mercurials as a diuretic in cardiac, renal and other problems of fluid storage. We are all particularly indebted to Dr. Keith and his co-workers[6] for combining the use of a controlled ionic diet with the administration of ammonium chloride or ammonium nitrate as great adjuncts to mercurial diuretics. The experimental demonstration of the role of sodium and potassium in the storage and clearance of body fluids by Barker[7] lead to the successful application of these findings as a more physiologic control of clinical edema. Certainly, this liberal diet which is low in sodium, high in potassium with potassium chloride as a salt substitute may be well tolerated by chronically ill patients for long periods of time.[8] More recently Keith and Binger have become interested in the diuretic effects of potassium and they have shown potassium nitrate to be an efficient diuretic salt.[9] It is interesting that all of the above principles have been in use in one way or another for many years, but only in the last few years have sufficient studies been made to correlate the whole into quite an efficient diuretic regime. I regard the development of such a flexible program of diet, minerals and diuretics as one of the finest steps made for the comfort of the patient carrying edema and all that its control connotes in combating the underlying process.

It must be remembered that edema is only a symptom and that the fundamental problem must not be slighted. The volume of fluid in the edematous patient is frequently much larger than one's greatest estimate. This fluid must be cleared through the kidneys and often either the long standing passive congestion or actual renal vascular disease or both alter greatly

From the Bettie Soper Clements Ward of Passavant Memorial Hospital and the Department Experimental Medicine of Northwestern University Medical School.

Read before Section on Medicine of Illinois State Medical Society, at Peoria, May 18, 1937.

their ability to clear minerals and waste products of protein metabolism. No doubt nature's dilution of retained materials is often a most important physiologic safeguard. The reverse of the process, therefore becomes a most important

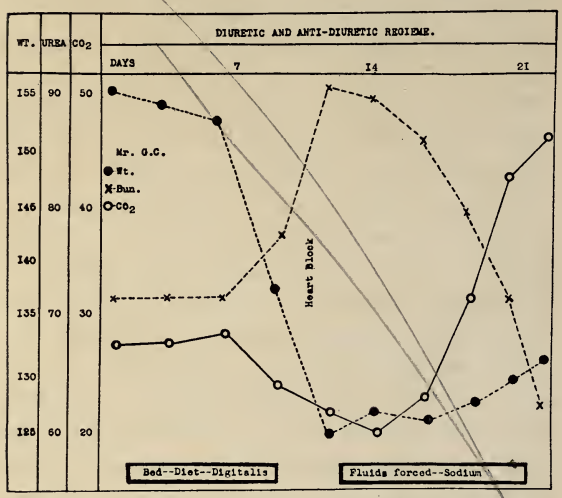

Figure 1. Note the rapid rise in the blood urea nitrogen and the fall in the CO_2 following the dieuresis. Heart block was associated with the concentration of digitalis in the edema fluid. An active anti-diuretic regime consisting of forcing fluids and giving sodium resulted in correcting the uremic manifestations as described in the protocal.

matter to such patients unless careful observation

The following three cases serve to show some of the dangers experienced in rapid diuresis. is maintained.

G. C. male, aged 34 years, complained of shortness of breath, nocturia, edema and ascites. The chief physical findings were blood pressure 158/128, pulse 90, regular, large heart, systolic blowing murmur at the apex, signs of fluid at the right subscapular region, liver down a hands-breadth, moderate ascites, and moderate edema of the sacrum and lower extremities. The chief laboratory findings were R. B. C. 5,370,000, Hgb. 12.0 grams, W. B. C. 14,100, urine +++ albumin, numerous hyaline and occasional granular casts. The phenolsulphonphthalein excretion was 4% for two hours and this was verified by repeated examination. The urea clearance was 10% of normal. The blood urea nitrogen was 73 mgms. and the carbon dioxide combining power was 26.8 volume %. A low sodium-high potassium neutral ash diet was started and digitalis I. B. D. was prescribed. Potassium chloride was used as a salt substitute. Reference to Chart I clearly shows the course which followed.

GRAPH I

As diuresis began and the weight began to fall, there was a concentration of the blood urea nitrogen from 73 to 91 mgms. and the carbon dioxide fell from 26.8 to 20.0 volume %. Headache, nausea, and orthopnea appeared. An antidiuretic regieme was initiated which consisted of stopping the digitalis, increasing the fluid intake from 1500 to 2500 c.c. per day and the addition of calcium carbonate 10 grams and sodium bicarbonate 5 grams daily. The diuresis continued for four days and the body weight continued to drop. A short period of coupling was followed by complete heart block (pulse 45). A rapid improvement followed the forcing of fluids, alkalinization and a period of balanced intake and excretion of fluid.

This case demonstrates the concentration of nitrogenous products, the deepening of the acidosis and heart block associated with a rapid diuresis. An active antidiuretic plan of stopping digitalis and potassium chloride and the addition of sodium bicarbonate and increased fluid intake, arrested the diuresis. Body weight increased slightly and there was a relief of symptoms. A return to the diuretic plan resulted in a complete loss of edema and a marked improvement of the cardiorenal state.

Mr. F. J., manufacturer, aged 50 years, a known severe hypertensive for years, complained of shortness of breath, weakness and periodic edema of the legs for one and one-half years. Headaches, nausea, marked edema, ascites and nocturnal dyspnea, for which he had entered two hospitals for the preceding eight months, were the chief presenting complaints. There was marked enlargement of the heart, rate 110, with a pronounced gallup rhythm. He was very stuperous and a uremic odor was noted. Blood pressure was 250 to

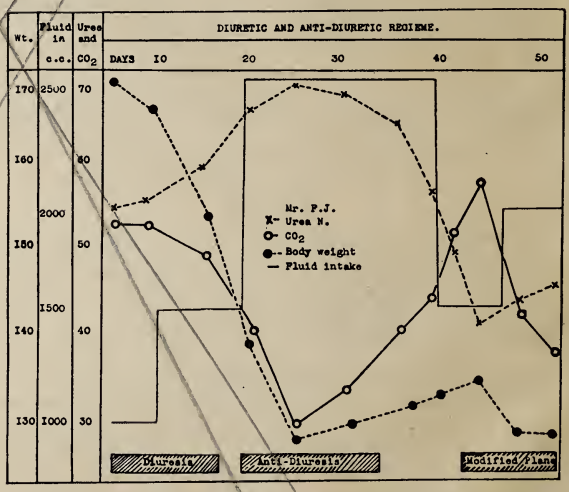

Figure 2. A rapid rise in blood urea nitrogen and a decrease in the CO_2 with uremic stupor following the dieuresis. Note the decrease in body weight. An antidiuretic regime of forcing fluids, change of diet to alkaline-ash type with the addition of alkaline salts corrected the acidosis and the uremic state. The patient was rehabilitated by a modified dieuretic program as described in his protocal.

260 over 140 to 156. The urine specific gravity was 1.012 and it contained 1 gram of albumin per liter. A moderate number of hyalin and occasional coarse granular casts were present. The phenolsulphonphthalein excretion was 4% and checked at 6% for two hours. Other laboratory findings were total serum proteins 7.94, serum albumin 4.46, R. B. C. 4,330,000, HGB 11.2